

needed to increase the cure rate. In vitro experiments show strong antiviral effects of fluvastatin against HCV.

Aim of the study: To assess the clinical outcome of fluvastatin addition to the standard regimen for treatment of chronic HCV in Egypt.

Subjects and Methods: The study included 80 patients with chronic hepatitis C virus infection fulfilled clinical, laboratory and histo-pathological criteria to be ready for interferon therapy, divided into two groups: Group I (n=40) received standard treatment for HCV (pegylated interferon and ribavirin) and group II (n=40) received standard treatment plus fluvastatin (80 mg/daily). Before and after 6 months of treatment liver function tests and HCV-RNA were evaluated.

Results: Addition of fluvastatin to the standard HCV treatment (pegylated interferon and ribavirin) significantly increased SVR from (55% to 62.5%; $P < 0.01$) and significantly decreased viral load in relapser patients ($P < 0.001$). No significant differences and correlations were found between serum levels of LDL-cholesterol and viral load before and after treatment in both groups.

Conclusion: Fluvastatin can be used to increase SVR when added to standard treatment (pegylated interferon and ribavirin) of chronic HCV.

OL-003 Genetic changes in the interferon sensitivity determining region of hepatitis C virus during the natural course of chronic hepatitis C 3a may lead to non-response to interferon therapy

A. Raza^{1*}, J. Asad², N. Zaman², H. Aziz¹, S. Murtaza¹, J. Irfan¹. ¹Nuclear Medicine Oncology and Radiotherapy Institute, ²PMAS Arid Agriculture University Rawalpindi, Pakistan

Background: We have found 70% response rate in Pakistan for Genotype-3a patients with combination therapy of interferon and ribavirin. There is need to individualize the treatment to minimize the side effects.

Methods: For the current study five G3 patients, nonresponder of 24 weeks interferon-alpha-2b plus ribivirin therapy, with almost equal levels of viremia (1.8×10^7 IU/ml) at the end of treatment were selected. Mean viral load at week 36 was 2.6×10^6 IU/ml, at week 48 raised to 2.0×10^7 IU/ml. All samples were quantified on RotorGene3000™. Interferon sensitivity-determining region (ISDR) in non-structural region-5A was studied to link any genetic changes in virus genome with therapy resistance. ISDR was amplified using genotype 3a specific ISDR primers followed by sequencing and bioinformatics tools.

Table 1. Mutations in Interferon sensitivity determination region of responder (R) and non responder (NR) Genotype 3a cases

R	CCGTCGTTGAAGGCCACTTG-CGGAACGCAT-TGGCCTCATCTAGACACTGAGCTAGTGG	58
NR		
R	CCGTCATTGAAGGCCCTCTGCCGG-ACGCCCTAGGCC-CCTCCAGACGCTGAGCTAGTGG	58
R	ATGCCAACTTGTTGTGGCGCAAGAGATGGGAGCAACATCACACGGGTAGAGTCTGAGA	118
NR		
R	ACGCCAACTTGTTGTGGCGCAAGAGATGGGAGTAAACATCACACGGGTAGAACTCTGAAA	118
R	CAAAAGTTGTGATCCTTGAATTCATTCGAACCTCTGAGAG	157
NR		
NR	CAAAAGTTGTGATCCTTGAATTCATTCGAACCACTGAGAG	157

Sequence homology between the responder (Naive) and Non responders was found with a Score of 181 bits (103), Identities = 141/159 (88%), Gaps = 4/159 (2%)

R	PSLKATCGTHMPLDTELVDANLLMRQEMGSNITRVESETKVVLDSFEPLR
NR	PSLKASCRTPQAPDAELVDANLLMRQEMGSNITRVESETKVVLDSFEPLR
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Amino acid comparison of responder and non responder with 84.6% identity in 52 amino acid residues overlap; Score: 212.0; Gap frequency: 0.0%

Result: A157bp ISDR product corresponding to 52 amino acid protein, when compared with responder HCV patient naïve genome [Table 1], eight amino acid mutations were detected including deletions and substitutions affecting the molecular weight of protein. Gly, Trp, Pro, Leu, His,

Thr were substituted by Arg, Gln, Ala, Pro, Pro, Ala respectively.

Conclusion: Mutations in ISDR region may have role in virus-resistance and high viremia, influencing the therapy response. Screening will help in deciding treatment plan making it cost-effective.

OL-004 Sequence analysis of hepatitis C virus 5' non-coding region

L. Zhang^{1*}, C. Qin², Y. An¹, X. Zhang¹, L. Wang³. ¹Department of liver Diseases, Jinan Infectious Disease Hospital affiliated to Shandong University, Jinan, Shandong province, 250021, PR China, ²Department of Digestive Diseases, Provincial Hospital affiliated to Shandong University, Jinan, Shandong Province, 250021, PR China, ³Central Laboratory, Jinan Infectious Disease Hospital affiliated to Shandong University, Jinan, Shandong province, 250021, PR China

Objectives: Our aim was to identify the prevalence of hepatitis C virus (HCV) genotype and investigate genetic mutation of HCV 5' non-coding region (5'NCR) sequences in Shandong province of China.

Methods: Serum samples from 118 chronic hepatitis C patients hospitalized in Jinan Infectious Disease hospital were collected. Serum samples were amplified from 5'NCR by RT-PCR and PCR products were sequenced by Sangon Biotech (Shanghai) Co., Ltd. Sequences of 5'NCR of the patients were compared with reference HCV strains from Genbank and phylogenetic tree analysis was performed.

Results: The cases among genotype 1b, 2a, 1a, 3a, 3b, 6a were 65 (55.1%), 45 (38.1%), 2, 1, 2 and 3, respectively. Sequences of 5'NCR in 42 genotype 1b patients were identical. Compared to reference HCV 1b strains, 23 genotype 1b patients have 1-2 bases mutation with two characteristic nucleotide mutation sites (120 C-T and 204 C-T). The homology of 5'NCR among genotype 2a patients was 97.8%-100% with characteristic nucleotide mutation sites (site 222 C-T and 247 C-T). The homology of genotype 1a, 3a, 3b and 6a with the same genotype HCV strains from Genbank was high, only with 1-3 bases mutation.

Conclusions: The predominant genotype of chronic hepatitis C patients in Shandong province is 1b, followed by 2a and a small amount of 1a, 3a, 3b, 6a. Genotype 3a and 6a are not reported before. Sequence of HCV 5'NCR is highly conservative and accords to HCV strains worldwide. Both genotype 1b and 2a have characteristic nucleotide mutations.

OL-005 A higher correlation of HCV core antigen with CD4+ T cell counts in HCV/HIV coinfecting patients

T. Shen¹, X.M. Chen¹, W.D. Zhang², Y.L. Xi², G.H. Cao³, Y.H. Zhi⁴, S.W. Wang⁴, L. Wei⁵, H. Zhuang¹, F.M. Lu^{1*}. ¹Infection Disease Center & Department of Microbiology, Peking University Health Science Center, Beijing 100191, China, ²Department of Epidemiology, College of Public Health, Zhengzhou University, Henan, China, ³Shanghai County People's Hospital, Shanghai, Henan, China, ⁴Shanghai Center for Disease Control and Prevention, Henan, China, ⁵Institute of Hepatology, Peking University People's Hospital, Beijing, China

Background: Development of HCV infection is typically followed by chronic hepatitis C (CHC) in most patients, while spontaneous HCV viral clearance (SVC) occurs in only a minority of subjects. With the development of techniques for direct detection of the HCV virus (RNA or core protein), it is expected that HCV infectious status can be evaluated better if the results of HCV antibodies and virus detection were